DISSERTATION ON

A STUDY OF MANAGEMENT OF ACUTE ELECTRICAL BURNS OF UPPER LIMBS

Submitted for

Partial fulfillment of

MCH. BRANCH – XVI

(PLASTIC SURGERY)

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI – TAMILANDU

AUGUST 2008

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF MANAGEMENT OF ACUTE ELECTRICAL BURNS OF UPPER LIMBS"** is a bonafide work done by **Dr.G.SENTHIL, MCh.** (Plastic Surgery), Kilpauk Medical College, Chennai – 10 under my guidance and supervision.

Prof. Dr. S.R.Vijayalakshmi, MCh.
(Plastic Surgery)
Head of the Department
Professor of Plastic Surgery
Govt. Royapettah Hospital
Kilpauk Medical College
Chennai – 600 010.

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF MANAGEMENT OF ACUTE ELECTRICAL BURNS OF UPPER LIMBS"** is a bonafide work done by **Dr.G.SENTHIL, MCh.** (Plastic Surgery), Kilpauk Medical College, Chennai – 10 in partial fulfillment of the regulation for M.Ch. (Plastic Surgery) degree examination of the TamilNadu Dr.M.G.R. Medical University to be held in August, 2008.

Prof. Dr. S.R.Vijayalakshmi, MCh.
(Plastic Surgery)
Head of the Department
Professor of Plastic Surgery
Govt. Royapettah Hospital
Kilpauk Medical College
Chennai – 600 010.

Dr. DHANAPAL, MD, DM,
(Cardiology)
Dean
Kilpauk Medical College
Chennai – 600 010.

ACKNOWLEDGEMENT

I am extremely thankful to the dean **Prof.Dr.DHANAPAL, M.D., D.M.,** (cardiology), Kilpauk Medical College for permitting me to utilize the clinical materials of this hospital.

I am very grateful to our professor and head of the department of plastic surgery, **Prof.Dr.S.R.VIJAYALAKSHMI, M.Ch (Plastic Surgery)** for her constant encouragement and unfailing support which gave me the confidence to do this project.

I also express my sincere gratitude to **Prof.Dr.MADHIVANAN, M.Ch (Plastic Surgery)** for his valuable suggestions in preparing this dissertation.

I express my sincere thanks **to Dr.DHANICKACHALAM, MCh (Plastic Surgery),** Professor of Plastic Surgery for his valuable suggestions and kind help in this study.

I thank all the Professors and Asst. Professors of the Department of Plastic Surgery, Kilpauk Medical College for their support during this study.

I acknowledge with thanks for the ready help rendered to me by other medical and paramedical staff.

I thank all the patients whose co-operation enabled me to complete this study.

CONTENTS

INTRODUCTION

Electrical burn is a unique form of trauma, in which mortality and morbidity are very high when compared to thermal burns.

The effects of electrical current depend on the type of current, voltage, tissue resistance, the pathway and the duration.[1]

Injuries caused by exposure to 1000 volts or greater are considered high tension electrical burns. High tension electrical current may cause 'flash' burns, 'True' electrical burns or secondary thermal burns.[2]

High-tension electrical burns results in cutaneous injuries, severe damage to underlying muscles, nerves, blood vessels and bones.

Every organ system can be injured by the passage of current. **Respiratory arrest, cardiac arrest, ventricular fibrillation, renal failure, gangrene** of the extremities are some of the early life threatening complications.

Upper limb involvement is present in majority of the electrical burn injuries. Upper limbs may have entry point, exit point or both. Injury may range from simple flash burns or low voltage contact burns to devastating gangrene of the limbs.

In this study, efficacy of **fasciotomy** in salvaging upper limbs, adequate debridement with early flap cover using distant flaps in healing of post electric burn wounds and amputation of gangrenous parts of upper limbs were assessed.

Electrical burns involving upper limbs are treated by cleaning the wound. Fasciotomy is done in circumferential and deep burns to prevent increase in compartmental pressure.

In high tension electrical burns involving upper limbs, where tissue destruction is massive with ischemic gangrene, **amputation** is performed to reduce the myoglobin and other toxic metabolite load and to reduce infective complications and the risk of secondary hemorrhage.

All the efforts are taken to salvage the limb with adequate debridement and early flap cover using various **distant flaps**. Local flaps are avoided because of the unreliable vascularity due to extent of the electrical injury to the adjacent areas.

Early split thickness skin grafts for remaining raw areas occurring due to flash burns are also gaining importance since repeated excision and early cover to prevent deepening of the wound following infection.

Even third degree burns are excised starting from third post burn day and early split thickness skin graft is done to reduce the morbidity.

In case of **lightning**, the commonest cause of death is respiratory arrest. So, if only artificial respiration is started immediately, patient can be revived. Since this should be done at the site of accident, which is more often remote, the mortality rate remains high.

AIMS AND OBJECTIVES

1. To study the **progressive nature** of electrical burn injury in upper limbs.

2. To assess the efficacy of **fasciotomy** as a limb salvage procedure.

3. To analyse the **timing of wound debridement and flap cover** in the final outcome.

4. To compare our results with studies from other standard institutions

REVIEW OF LITERATURE

EPIDEMIOLOGY

Since its introduction for public use, electricity has been one of the most potentially dangerous commodities in our society.[3]

- Electrical injuries are more common in healthy young males.
- Upper extremity involvement is seen in most of the electrical burns victims.
- Mortality is high in high-tension electrical burns.

Properties of Electricity

An understanding of some fundamental laws of physics is an essential prerequisite of proper management.

Electricity is the flow of electrons from atom to atom.[4]

Amperage is the term used for the rate of flow of electrons.

Voltage is a measure of potential energy of an electrical field to cause an electrical current in an electrical conductor.

Resistance is measured by the unit termed **ohm**. The resistance offered to the flow of electricity by any tissue is directly proportional to its length and inversely proportional to its cross sectional area.

Ohm's law states that amount of current flow is directly proportional to voltage and inversely related to resistance.[7]

Current (I) = Voltage (E) / Resistance (R)

Passage of electric current through a solid conductor results in conversion of electric energy into heat, the Joule effect.

Heat production in joules is proportional to the power dissipated, multiplied by the duration of contact and is expressed by the equation.[8]

$J = I R T.$

Resistance of the human body has been likened to that of a leather bag filled with an electrolyte fluid, with high resistance on the outside and lower inside except bone.[1]

Skin resistance also varies depending on moisture content, thickness and cleanliness.

Resistance offered by the callused palm is 1,000,000 ohms/cm^2, but the average resistance of dry normal skin is 5000 ohm/cm^2. This resistance may decrease to 1000 ohms/cm^2 if hands are wet.[6]

Electric arc

Contact with high-voltage current may be associated with an arc or light flash. The arc has an intense pale violet light consisting of ionized particles producing temperature as high as 4000°C. The electric arc remains the cause of most high voltage electrical burn injuries.

Effects of electricity on the body

Effects of electricity in the body is determined by various factors.[9]

1. Amount of current :

1 mA current for 1 second contact time is the threshold of perception.

10 – 15 mA causes sustained muscle contraction

50 – 100 mA results in respiratory paralysis and ventricular fibrillation

2. Type of current

Alternating current produces more damage than the direct current.

3. Pathway of current

A pathway of current from hand to hand (across the heart), is associated with ventricular fibrillation and high mortality.

4. Duration of contact

Electrical injury is directly proportionate to contact duration.

Resistance and voltage are the other factors which affect the electrical injury on the body.

PATHOPHYSIOLOGY

Electricity involves the flow of electrons along the path of least resistance toward a natural ground. Children's thin skin and high water content results in less resistance, compared with adults. Skin, bone, tendons and fat provide the most resistance to electrical current.[10]

Nerves, blood vessels, mucus membranes and muscles are the best conductors.[10]

Cross-sectional area is inversely proportional to tissue damage. Therefore, small areas such as joints receive maximal injury. **Vertical pathway** (head to toe) of current is more dangerous than a **horizontal** (hand to hand) **pathway** as the former has more damage on the central nervous system.[11]

Alternating current produces **tetany** and the **locked-on** phenomenon. Although tetany occurs in all muscles that are stimulated, flexor groups are usually stronger and predominate. Individual's grasp in locked-on position increases the duration of the electrical contact.[12]

Heat generated on the vessels causes coagulation and occlusion. Electrical injury to the vascular wall produces **delayed thrombosis** and **bleeding.**

When current passes through large vessels at its entrance or exit points, it may cause a pronounced inflammatory reaction as well as small areas of vascular wall necrosis. The latter may lead to immediate or delayed rupture of the vessel. Current flowing through larger vessels that are located beyond the entrance and exit points of the current may damage the vessel, precipitating the development of mural thrombi, yet vessels usually remain patent. Flow through these larger vessels

dissipates heat and limits damage to their walls. In smaller vessels, this heat injury results in vessel thrombosis.[13]

COMPARTMENT SYNDROME

Compartment syndrome is a limb-threatening and life-threatening condition observed when perfusion pressure falls below tissue pressure in a closed anatomic space.

Compartment syndrome may develop as a result of acute ischemic insult to the musculature. At this point, blood flow through the capillaries stops. In the absence of flow, oxygen delivery stops. **Hypoxic injury** causes cells to release vasoactive substances (eg, histamine, serotonin), which increase endothelial permeability. Capillaries allow continued fluid loss, which increases tissue pressure and advances injury. Nerve conduction slows, tissue pH falls due to anaerobic metabolism, surrounding tissue suffers further damage, and muscle tissue suffers necrosis, releasing myoglobin. The end result is loss of the extremity and, possibly, the loss of life .[14]

Normal interstitial pressure is 4-6 mm Hg. However, rising interstitial pressure overwhelms perfusion pressure.As intracompartmental pressure rises, venous pressure rises. When venous

pressure is higher than capillary perfusion pressure, capillaries collapse. Intracompartmental pressures greater than 30 mm Hg are generally agreed to require intervention in the form of fasciotomy and decompression.

Neurological dysfunction is present in some form, virtually in all patients due to transient nerve injuries resulting in numbness and tingling in most cases.

Mass depolarisation of the brain may lead to a loss of consciousness, amnesia and coma. Spinal card involvement may result in transverse myelitis. Transverse myelitis may have delayed onset with poor prognosis.Peripheral nerve lesions can appear later. The commonest presentation is **foot drop.**[15]

THEORIES ON TISSUE DAMAGE

1. Heat production by tissue resistance

As the current passes in the body, heat is produced proportionate to the tissue resistance.

Tissue resistance progressively increases from nerve to blood vessels, muscles, skin, tendon, fat & bone.

Bone, having the greatest resistance, generates more heat as the current passes resulting in deep periosseous tissue destruction.[16]

2. Progressive muscle necrosis

The majority of current would preferentially travel along the lines of lesser resistance, particularly the blood vessels.

These vessels are injured leading to delayed arterial occlusion and progressive ischemic necrosis.[17]

Endothelial cell damage contributes to vessel thrombosis, further ischemia and tissue necrosis.

3. Cell membrane lysis by electroporation

Electrical injury produces a degree of cell shock. Intracellular ATP is changed to AMP. Sodium pump becomes ineffective. As a part of immediate cell injury, proteases are released. This can trigger coagulation cascade and complement degradation.[18]

4. Arachidonic acid metabolites causing injury

Electrical injury arbitrates phospholipase A at cellular levels. Phospholipase A, cleaves the phospholipids that are bound to cholesterol and triglycerides in the cell membrane. This results in release of arachidonic acid metabolites, such as thromboxane.[19]

In 1984, **Robson**, using rat model, reported a decrease in tissue damage with electrical injury where topical therapy was used to block the formation of thromboxane.

HISTORY WHICH SHOULD BE OBTAINED FROM THE PATIENT / RELATIVES

- **Voltage and type of current :**

  - Standard household current in India is 220 – 250 Volts AC.

  - Power lines and electrical transformers constitute high voltage energies greater than 1000 V.

- **Length of exposure :**

- It is important to know whether the patient had a brief contact or sustained (tetany) contact

- conditions associated with electrical injury such as **wet skin** came in contact with a live wire or the victim had bath in a **bath tub** at the time of electrical injury are asked to know the gravity of the situation.

- History of **pre-existing medical problems.**

- History of **cardiac arrest or fall from the height** is enquired.

CLINICAL EXAMINATION

- Airway, breathing and circulation and conscious level of the patient is checked.

Circulation is checked by, skin colour, temperature capillary refill urinary output and heart rate.

Movement of all four limbs, skeletal injuries , concealed injuries like chest and intra abdominal injuries should be checked.

UPPER EXTREMITIES

The loss of an arm or leg is one of the most devastating consequences of a burn injury. Amputation rate as a result of high-voltage electric burn injury is high. Deep thermal burns complicated by extensive soft tissue necrosis or invasive infection may produce non-salvageable extremities. Advances in reconstructive surgery have resulted in decreased amputation rates after electrical burn injuries. Traditionally, amputation after burn trauma has been related primarily to high-voltage electric injury. These amputations may be isolated to a major extremity or to digits, hands, or feet.[1]

Electrical injuries may be associated with fall from a height. Tetany may cause fractures or dislocation. Entry and exit burns to be looked for. Flash burns may appear identical to standard thermal burns.

Circumferential burns, increasing pain especially on passive motion in association with tight forearm are indicative of compartment syndrome .Presence of distal palpable pulse doesn't rule out the compartment syndrome, as the capillary perfusion may be absent.[20]

Electrical burns injuries may involve peripheral nerves, gastrointestinal system, cardiovascular system and respiratory system.

INVESTIGATIONS

LAB STUDIES

Hemoglobinemia may appear after electrical unjury as a result of lysis of red blood cells.Raised renal parameters may indicate pre-renal (inadequate I V fluids) or renal failure due to deposition of toxic myoglobin in the renal tubules. Hemoglobin and myoglobin may be present in urine due to extensive rhabdomyolysis and lysis of red blood cells.

Complete-blood count, electrolyte levels, glucose, urea, creatinine. Glucose, urea, creatinine and urine analysis are the baseline blood investications.

Increased serum creatinine and creatine phosphokinase levels can occur due to progressive muscle necrosis. Damage to the Cardiac muscles may be reflected by raised cardiac enzymes such as LDH, SGOT and AST (Aspartate amino transferase).

Imaging studies and other studies

- Chest radiograph is taken to rule out hemo-pneumothorax.
- 12 lead ECG is taken in all the patients. Cardiac monitoring may be reserved for patients with a history of cardiac arrest or loss of consciousness, dysrhythmia or abnormal ECG findings.

Identification of degree of skeletal muscle damage is most difficult in electrical injuries. There are some imaging studies which are proved to be useful.

- **Technetium Tc 99m stannous pyrophosphate scintigraphy**[21] Technetium Tc 99m stannous pyrophosphate (99m Tc-PYP) muscle scan is a sensitive and reliable diagnostic tool to define the extent and location of muscle injury. This test can be performed in most hospitals with a nuclear medicine department. An isotope is infused intravenously, and the scan is performed 2 hours later. Several characteristic scintigraphic imaging patterns indicate muscle damage. An increased cellular uptake of 99m Tc-PYP (hot spot) identifies an area of muscle damage consisting of 20-80% viable muscle, which should be followed clinically. Conversely, areas with no uptake of this radioactive material (cold spot) are devoid of blood supply and obviously necrotic.

- **Nitroblue tetrazolium enzyme** mapping is useful to identify viable and non-viable tissues.

- **Microscopy** as a guide to primary excision is the most accurate diagnostic procedure for muscle viability. In this method, **frozen section** histology is used to determine muscle damage. Because muscle cross striations may be preserved in dead or dying muscle, morphology of the

muscle cell nuclei is the best indicator of viability. Muscle containing clumped nuclear chromatin or poor staining with hematoxylin indicates myonecrosis and should be excised.

- **Xenon^{133} washout technique** is useful to assess the extend of muscle damage by measuring muscle blood flow.

However, **accurate pre-operative assessment, clinical and surgical judgement** of the surgeon is most important.

MANAGEMENT OF ACUTE ELECTRICAL BURNS

MEDICAL

Airway : Patency of the airway is maintained.

Breathing (**ventilatory support**) : High voltage electrical injuries may be associated with respiratory arrest,life threatening rhythms or multi systemic involvement requiring ventilator support.

Circulatory support :

Intravenous access, possibly central venous access is indicated depending upon the severity of the injury. Accurately estimating extend of tissue damage in high voltage injury is difficult as it involves larger amount and depth of tissue injury.

Ringer lactate solution is started as 7ml /% of burns /kg body weight. Fluid flow is adjusted to maintain a minimum urine output of 1 ml/kg/hr.[22] Blood transfusions were given in cases of anemia due to red cell destruction.

Renal function

Patients with high voltage injuries are susceptible to rhabdomyolysis and myoglobinuria. Myoglobin is toxic to kidney

because it may crystallize, occlude urine flow and result is acute renal failure.[23]

Adequate hydration is recommended. Urine output is maintained at the rate of 1 – 1.5 ml/kg/hr.

SURGICAL MANAGEMENT

i) FASCIOTOMY

Normal cellular metabolism requires 5-7 mm Hg oxygen tension; this is easily maintained with the capillary perfusion pressure averaging 25 mm Hg and interstitial pressure 4-6 mm Hg. However, rising interstitial pressure overwhelms perfusion pressure.

Electrical burns resulting in tissue hypoxia initiates inflammatory response by releasing vaso active amines (histamine, serotonin) which potentiates edema and increasing intra-compartmental pressure this in turn raises venous pressure. When venous pressure is higher than capillary perfusion pressure, capillaries collapse. The intra-compartmental pressures greater than 30 mm Hg need fasciotomy and decompression. Affected extremity is not elevated. Keeping extremities level with the body decreases limb mean arterial pressure without changing intra-compartmental pressure. After an elevation of 35 cm, a

decrease in the mean arterial perfusion pressure of 23 mm Hg and no change in intracompartmental pressure.

ANTERIOR COMPARTMENT OF THE FOREARM

To release the anterior compartment 'S'-shaped incision extends from medial biceps region to curving again over the ulnar aspect of the wrist and extending to palm.[25]

POSTERIR COMPARTMENT OF THE FOREARM

Dorsal incision from lateral epicondyle to midline of wrist is made to decopress extensor muscles

AT THE WRIST LEVEL

Carpal tunnel is decompressed to free the median nerve. Soft tissue flap is used to cover the median nerve at the wrist.

INCISIONS PLACED OVER THE HAND TO RELEASE COMPARTMENTAL PRESSURE

Two longitudinal incisions were placed over the second and the fourth metacarpal bone, to release the dorsal and volar interosseous muscle compartments.

Thenar and hypothenar muscle compartments were released by incisions over the radial and ulnar surface of the first and fifth metacarpals.

INCISION OVER THE FINGERS :

With severe swelling of the fingers finger fasciotomy is performed over the ulnar border of the index, middle and the ring fingers and radial borders of the thumb and little fingers to preserve the protective sensation of the fingers.[26]

PHYSIOTHERAPY AND SPLINTING

Physical and occupational therapy is instituted early to maximize hand function.

Hand is splinted in functional position with the wrist in 30 degrees of extension, metacarpophalangeal joint in 90 degees flexion, interphalangeal joint in extension and thumb in abduction.

WOUND DEBRIDEMENT

In high-voltage injury, necrotic tissue may extend beneath what appears to be normal uninjured skin and subcutaneous tissue.

Adequate wound debridement is the key to success of wound management. A proper wound debridement is removal of all devitalized tissues and retaining viable tissues.

Ponten et al[27] reported that this coagulation necrosis may exist in the contiguous deeper tissues as far as 25 cm from the point of entry and exit.

It is useful to observe and document the viability of the muscle at the time of initial fasciotomy.

Ability of the muscle to bleed and contract, is the most reliable clinical indicator of muscle viability.[28]

Tecnetium-99m pyrophosphate muscle scans may be useful in assessing 'cold' areas of irreversibly damage muscles and 'Hot' areas of 20-80% necrosed muscle fiber.

Frozen sections at the time of debridement to assess the viability of the muscle are useful.

Clayton et al showed that muscle viability can be assessed by directly measuring the blood flow by $Xenon^{133}$ washout technique.[29]

Obviously necrotic tissue should be removed as early as possible, preferably at the time of fasciotomy.

Definitive flap cover or skin graft is not advisable within 48-72 hrs of injury, as the ischaemic insult at the cellular level is not complete before this period apart from unstable general condition of the patient. [30]

WOUND COVERAGE

After completing the definitive debridement, exposed normal tissue or minimally injured tissues should be covered so as to prevent further damage by desiccation. Desiccation alone actually stimulates the production of thromboxane.[31]

Split skin graft may be sufficient for flash burns wound.

Flap cover is required in high-voltage electrical injury as the wound is deeper and may expose nerves, vessels, tendons and even bone.

With electrical injury of larger areas, local flaps may not be available. Also, the extent of the electrical injury is unpredictable. So, the usage of local flaps is risky and is avoided.

Distant pedicled flaps in the form of axial groin flap and the superiorly based or the inferiorly based random abdominal flaps are

reliable choice of wound coverage. Appropriate flap design and positioning of the arm will reduce shoulder and elbow discomfort.[32]

Recently **microvascular anastomosis** using free flap is done in many centers. Microvascular anastamosis is a relative contraindication as vascular anastomosis should be done in uninjured vessel which may be well away from the original wound. **Vein grafts** to carry the anastomosis proximal to the zone of injury may be incorporated in the flap design.[33]

Distant pedicled flaps are technically easier and reliable choice of electrical burn wounds.[34]

MATERIALS AND METHODS

STUDY DESIGN AND PERIOD

This prospective study was conducted from September 2005 to March 2008, a period of 2½ years.

STUDY CENTRE

This study was conducted in Department of burns ,plastic and reconstructive surgery at kilpauk medical college hospital ,Chennai.

SUBJECT SELECTION

The inclusion criteria for the study were electrical burns involving upper limb but not exceeding 30% TBSA.Lightning injuries and electrical burns which involved more than 30% TBSA are excluded in this study.

A total number of 52 cases were included in this study. All age groups and both sexes were included in this study.

STUDY METHODS

Electrical burn injury is assessed at the time of admission. Extent of injury is marked in the case sheet. Photographs were taken for record

purpose. Over the days, progression of the injury is observed and recorded.

Fluid requirement is much greater than thermal burns. Ringer lactate is administered 7ml/kg body weight/% of electrical burns initially adjusted to maintain 1 – 1.5 ml/kg/hr urine output. Blood transfusions were given in cases of anemia due to red cell destruction.

Intravenous administration of sodabicarbonate and large volumes of intra venous fluids are useful in methemoglobinuria cases.

Upper limbs were observed for any signs of increased compartment pressure such as disproportionate pain, more pain on passive stretch, hard shiny skin over a swollen limb and absence of pulse or sensory deficit.

Keeping extremities level with the body increases tissue perfusion by decreasing limb mean arterial pressure without changing intra-compartmental pressure. Tight compressive dressing is avoided. Fasciotomy was done in circumferential deep burns to prevent increase in intra-compartmental pressure. Decompression was done in all muscle compartments. Nectrotic tissues were removed.

Volar incision medical to biceps tendon down to carpal tunnel and dorsal incision from lateral epicondyle to midline of wrist is made to decompress flexor and extensor muscles respectively.

In the hand, two longitudinal incisions were placed over the second and the fourth metacarpal bone, to release the dorsal and volar interosseous muscle compartments.

Thenar and hypothenar muscle compartments were released by incisions over the radial and ulnar surface of the first and fifth metacarpals.

Immediate decompression of tight muscle compartments with simultaneous radical debridement is done including immediate amputation of extremities that are clearly charred and non-salvageable.

Deep muscle compartments adjacent to the bone are also released. Muscle with doubtful viability is left for reevaluation at 48 – 72 hrs intervals.

After initial debridement, extension of tissue necrosis or gangrene is also recorded. Wound debridement is done with skin cover either by split skin graft or flap. Nerves and tendons are preserved even if they appear devitalised. Repeated debridements are performed to excise all

devitalised tissues. Definitive skin cover is given once the wound bed is healthy.

Skin cover is achieved by means of **split thickness skin graft** if the wound bed is healthy or granulating without exposing vital structures.

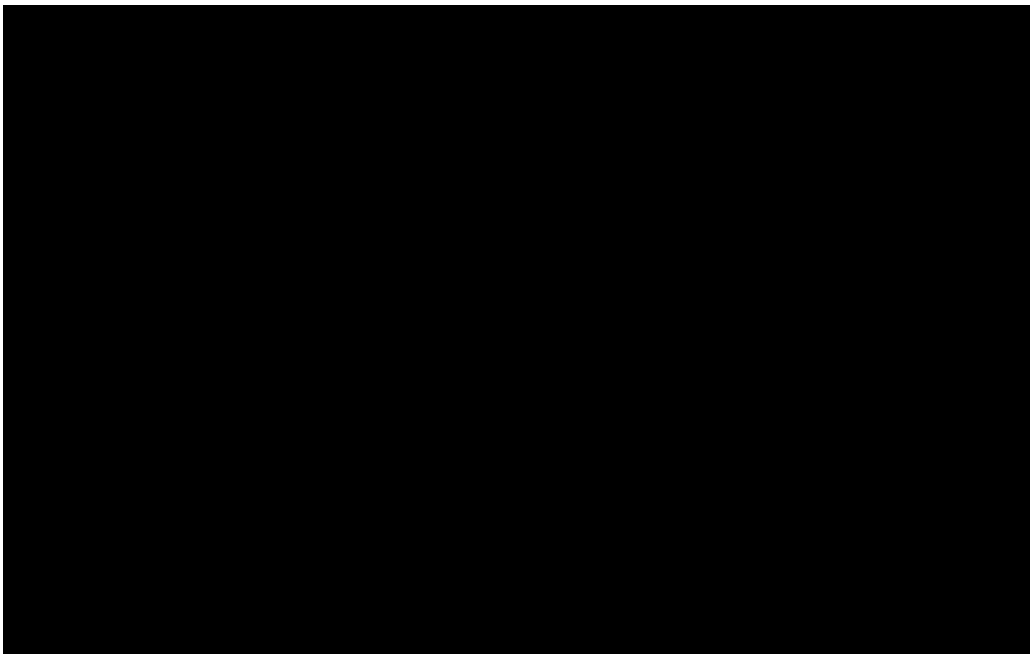
Exposed vessels, nerves, tendons and bone is covered with distant flaps. **Groin flap, abdominal flaps** either superiorly based or inferiorly based are commonly used distant flaps**. Reverse radial forearm flaps** and **posterior interosseous artery flaps** were also used in selected cases.

Amputation of the gangrenous part is done in established cases, to reduce myoglobin load , to prevent infective complications and to reduce the risk of secondary hemorrhages

RESULTS

I. Age Distribution

Involvement of the upperlimb electrical burns were seen more in adult and adolescent age groups (15 – 35 yrs).



II. Prevalence of Electrical burns

Total number of burns cases admitted in our hospital from September 2005 till March 2008, a period of 2½ years were 3747 cases.

- Total number of electrical burns cases admitted in the above period were 69 cases.
- Number of electrical burns cases included in this study were 52 cases.

- Prevalence of electrical burns in our burns unit was 2% of all burns admissions.

III. Upper Limb Involvement in Electrical Burns

- Total number of electrical burns admissions during my study period were 69 cases.
- Upper limb involvement was seen in 52 cases.
- Upper limb involvement in electrical burns injury was 74%.

IV. Sex Distribution

Total number of electrical burns cases included in my study were 52.

Males were the victim in majority of cases 51.

V. Etiology

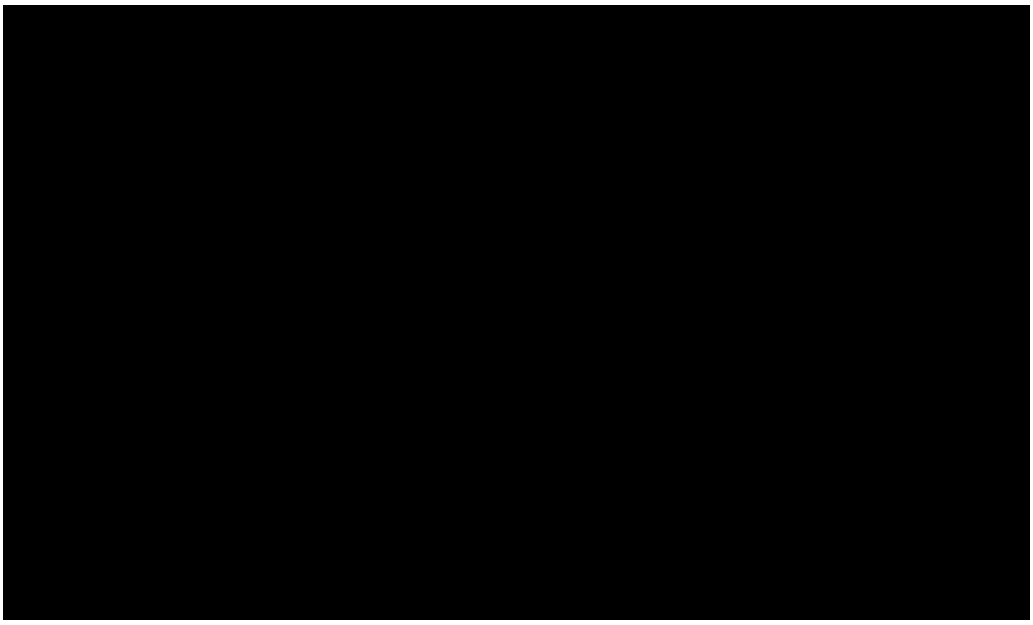
- History of accidental contact of the high tension live wires were seen in 20 cases.

- There were 17 cases of un-trained causal laborers who sustained electrical burns because of not adhering the safety measures during work. Five adolescent males had accidental electrical wire contact while playing cricket or retrieving kite. Ten cases were electrocuted when they were under the influence of alcohol.

Voltage

37 cases had high voltage (>1000 V) electrical burns.

15 cases had low voltage (<1000 V) electrical burns.



TIMING AND AREAS OF FASCIOTOMY

	I PBD	**II PBD**	**III PBD**
Foremarm alone	1	2	1
Forearm & wrist	1	1	
Hand alone		3	1
Foremarm, wrist & hand	2	2	

Out of 52 total cases in my study, fasciotomies were done in 15 cases.

In majority of the cases (13 cases) fasciotomies were done within 48 hrs of electrical burn injuries.

Of 15 fasciotomies, we could definitely save 6 limbs either by SSG or flaps. Of the remaining 9 cases the improvement of circulation helped

in minimizing distal amputations. In 4 cases required only disarticulation of fingers instead of below elbow amputations.

Amputation

Out of 52 cases, amputation at shoulder level was done in 1 case. Unilateral below elbow amputations were done in 5 cases. Above elbow amputations were done in 2 cases. Bilateral below elbow amputations were done in 2 cases. In 8 cases disarticulation of fingers were done due to gangrene of the fingers.

Of all cases one case developed secondary hemorrhage after guillotine amputation. It was done to reduce charred distal stump. It is obvious that it should have been done over normal tissue. Emergency exploration, ligature of the vessel more proximally and revision of amputation was done.

Of the 5 cases with unilateral amputations, 3 were potential candidate for bilateral amputations. Repeated debridement and flap cover saved at least one limb (2 dominant, one non-dominant) in those three patients.

AMPUTATIONS

Post electrical burn days	Disarticulation of fingers	Below Elbow		Above elbow	
		Unilateral	Bilateral		Disarticulation of shoulder
V		1			
VII	6	3	2	2	1
1-2 wks	2	1			
Total	8	5	2	2	1

SPLIT THICKNESS SKIN GRAFTS

Total number of cases : 52

SSG done : 19 cases

	V PBD		VII		1-2 wks		2-3 wks	
Total no. of cases			5		6		8	
	I	SE	I	SE	I	SE	I	SE
	0	0	2	3	3	3	0	8

SE – Serial excision

SERIAL EXCISIONS

Serial excisions were needed in 24 cases out of 52 cases. Progressive nature of injury was observed in electrical burn cases which required repeated debridements in 46% of cases.

FLAP COVER

Total number of cases flap done was 15 cases.

Flap type	V PBD	VII	1-2 wks	2-3 wks
Groin flap		2	3	1
Bilobed groin		1	1	
Superiorly based abdominal flap		1	1	1
Inferiorly based abdominal flap			1	1
Reverse radial forearm flap			1	
Posterior interosessious artery flap			1	

Single stage wound debridement and skin cover Vs Serial debridement and skin cover

	V PBD				VII PBD				1-2 weeks PBD				2-3 weeks PBD			
I wound debridement	10				25				17							
	A	S	C	F	A	S	C	F	A	S	C	F	A	S	C	F
	1		5		14	3	6	2	3	4	6	4				
Serial debridement					7				6				11			
	A	S	C	F	A	S	C	F	A	S	C	F	A	S	C	F
					0	2	3	2		2		4		8		3

A - Amputation
S - SSG
C - Collagen
F - Flap

DISCUSSION AND COMPARATIVE ANALYSIS

In our centre, upper limb involvement was seen in 74% of all electrical burns cases. Most of the cases bad entry points, some of them had exit points and few had both entry and exit burns.

12 Lead ECG was taken for all electrical burn cases and signs of myocardial injury or dysrhythmia were looked for. Continuous ECG monitoring was done in cases where 12 lead showed some abnormalities or when there was a history of cardiac arrest at the scene of accident.

A case of quadriplegia was admitted in our burns ward. This was due to fall from the lamp post after high tension electrical injury. However this case was not included in my study as he had 40% TBSA electrical burns.

A case of lightning burn was admitted in our burns ward. She had 45% TBSA lightning burns. She expired due to cardio-respiratory arrest.

The apparently seen cutaneous injury is the tip of an ice berg. As there will be an extensive destruction of all the underlying structures, fluid requirement is much greater than thermal burns.[36]

Ringer lactate is given as 7 ml/kg body weight/% of electrical burns for the first 24 hours. This fluid is then adjusted according to the urine output so as to maintain 1 – 1.5 ml/kg body weight/hour.

One of the major complication of high tension electrical injury is acute renal failure. Circulating hemoglobin is toxic to kidney and may lead to acute tubular nephropathy. Prompt fluid resuscitation is the key to prevent complications.[37] The use of IV fluids to increase renal perfusion and sodium bicarbonate to achieve urine alkalinity has been utilized in our burns unit. Blood transfusions were given to correct anaemia, which was due to massive red blood cells destruction due to electrical burns.

Limbs were observed for any signs of increased compartment pressure such as disproportionate pain, pain on passive stretch, hard shiny skin over a swollen limb and absence of pulse or sensory deficit.

Tight compressive dressing was avoided. Fasciotomy was done in all circumferential deep burns without waiting for any signs of increased compartmental pressure. Decompressions of all muscle compartments were done. Necrotic tissues were removed. This is done usually on the day of admission.

Amputation of the charred, severely damaged, non salvageable, gangrenous portion of the limbs were done.

In my study, disarticulation of fingers were done in 10 cases. Below elbow amputations were performed in 5 cases. Bilateral below elbow amputation was done in 2 cases. One patient required disarticulation of the shoulders joint as he developed gangrene of the whole upper limb upto axilla.

Clinical judgment of viable and nonviable tissues were made at the time of fasciotomies or wound debridements. Healthy muscle will appear bright brown in colour. It will bleed and contract when out. But unhealthy muscles will appear pale, lusterless and fail to contract. Unhealthy nerve will appear brownish yellow, dull or pale yellow or charred.

The exposed vital structures such as vessels, nerves and tendons were covered by local muscle adjustments or soft tissue flaps with skin graft or flap cover.

Deep muscles that were adjacent to the bone is also inspected.[41] Muscles with doubtful viability were left for re exploration and re-evaluation at 48-72 hours intervals.[42] Nerves and tendons were preserved even if they appear devitalized.

Repeated debridements were performed in order to excise all devitalized tissues and to reduce infective complications. Definite skin cover was provided after proper wound debridement in the form of split thickness skin grafts or flaps.

This was to reduce the mortality which associated with secondary hemorrhages. 2 units of compatible blood reserved for high risk patients. The wound was observed for secondary hemorrhage. In case of secondary hemorrhage from necrosed vessel, compression at the bleeding site; ligation of the bleeding vessel proximally where its wall is healthy to hold the ligature was done. This is a life-saving procedure in many of the electrical burns patients.

Skin coverage by means of autologous split thickness skin grafts were done in some patients over the healthy muscles or granulating surfaces. SSG does not give a stable skin cover for later intervention like tendon repair or nerve repair.[45]

So if a substantial number of exposed muscles, tendons and nerves are deemed viable at the initial or subsequent procedures, early coverage by a pedicled or free flaps may preserve essential functions in a way that no other method can achieve.

In my study, various pedicled distant flaps including Groin flaps, bilobed groin flaps, superiorly based abdominal flaps and inferiorly based abdominal flaps were used to cover the post electrical burn raw area with exposed vital structures in the upper limbs. Reverse radial forearm fascio-cutaneous flap and posterior interosseous flap were also used in my study.

Bilobed groin flap is based on a single pedicle which includes superficial circumflex iliac vessels and superficial inferior epiigastric vessels. This flap is very useful to cover the circumferential raw areas or defects around the wrist or the hand. In my study, bilobed groin flap was done in 2 cases.

Groin flap is a versatile, safe flap and technically easy to perform. 2 Groin flaps were done for osteoplastic thumb reconstruction in this study. First web space defect over the dorsum of the hand was covered by a groin flap in a case. 2 wrist defects were also covered by groin flaps.

Superiorly based abdominal flaps were used in 3 cases, one for the defect over the palmar aspect of the hand and another two cases were defects over the volar aspect of the wrist and distal forearm.

It is a random pattern flap raised in 1:1 ratio. Bulkiness of the flap can be thinned at a later date. It is reliable and easy to perform.

Inferiorly based abdominal flaps which include the superficial inferior epigastric vessels are reliable flaps for upper limb coverage. Defects over the dorsum of the hand and dorsal aspect of the forearm were covered using this flap.

Pedicled reverse radial forearm flap was used to cover a case of post electrical burns defect over the dorsum of the hand exposing tendons. This flap was planned after performing Allen's test to assess the patency of communication between the ulnar and radial arteries. As this patient was not willing for any distant flaps which required immobilisation of three weeks. Reverse radial forearm flap was chosen.

Posterior interosseous artery flap was used to cover a post electrical burn defect which exposed first metacarpal bone with neuro vascular bundle of a thumb. Doppler confirmation of anastomosis between the anterior interosseous artery with posterior interosseous artery at the level of 2.5 cm proximal to distal radio-ulnar joint was done before planning this flap. This flap provide a single stage repair, that reduced the hospital stay of the patient.

All flaps survived well.One flap showed partial skin loss which was subsequently grafted. Infection under the flap cover was noted in 3 cases, which was managed with antibiotics. Secondary hemorrhage due to sloughed out radial artery in a post burn wrist wound was managed by ligating the radial artery both distal and proximal over the normal appearing healthy vessel. 2 units of compatible blood transfusion were given for that patient. Patient was revived. Immediate flap cover using superiorly based abdominal flap was done.

Holliman et al,[39] pointed that early and repeated direct inspection of the damaged tissues is the most reliable method of assessing viability.

In this study, early wound debridement and skin cover in the form of SSG or flap cover was done in 13 cases; serial excisions followed by skin cover in the form of SSG or flap cover was done in 23 cases.

Parshley et al,[40] stressed the importance of immediate decompression of tight muscle compartments, to prevent further damage to the tissues and simultaneous radical debridement, including immediate amputation of the extremities that are clearly non-salvageable.

In this study unilateral below elbow amputations were performed in 5 cases; bilateral below elbow amputations were done in 2 cases;

disarticulation of fingers including 2 thumbs were done for 10 cases and disarticulation of shoulder joint was done in a case.

Kuzon et al and Zelt et al designed a model in a primate which showed that ischemia produced by an electrical current is complete by 72-96 hours. So, the wound coverage could be done within a week.

But in this study, progression of the injury was noted even after 5 days. So, repeated debridements were performed in order to excise all devitalized tissues; to reduce infective complications and to reduce the risk of secondary hemorrhages.

There are many **advantages** in early wound debridements and flap cover. Firstly, removal of the dead tissues reduces the toxic myoglobin in the circulation that may cause acute tubular nephropathy. Secondly, infections due to gram positive or gram negative organism is reduced by removing the non viable tissues which may act as a culture medium for the infective organisms.

Thirdly, the pungent or putrefying smell is reduced. Most of the patients will be anemic due to red blood cell destruction. They are encouraged to take more of oral intake of nutritious balanced diet. But the smell prevents them from eating. We have had cases where the care

givers (parents, spouse) refused to go near the patient because of the bad smell.

Fourthly, early wound cover either by SSG or flap is possible. It reduces the hospital stay and helps in faster rehabitation.

There are some **disadvantages** of early wound debridement and flap cover. The progressive nature of the injury has to be taken into consideration. The chances of exposing neurovascular bundle are high. The general condition may not be fit enough to permit major surgical procedures. The risk of secondary hemorrhage is more if the debridement inadequate or the injury is progressive. The flap loss is more when it was given in a progressive injury or over an inadequately debrided wound.

In this study, wound debridements were started on the fifth post electrical burn day. Although fasciotomies were done on the day of admission or on the second post burn day might be combined with excision of obviously non-viable or charred tissues.

Even though early wound debridement was done in most of the cases, we had to repeat the wound debridements in 30% of cases.

All the post electrical burn wounds were covered within 2-3 weeks either by flap/SSG. In the study group, the mean stay in the hospital was

18 days in contrast with patients who had delayed intervention for whom the hospital stay was prolonged (mean stay was 42 days).

One patient had secondary hemorrhage at the amputation stump, which was managed by ligating the vessel proximally and revising the amputation. One patient expired due to cardio-respiratory arrest on the day of admission. Another patient died of renal failure on fifth post electric burn day. Mortality rate greatly dropped after the fifth post electric burn day.

One patient has presented with gangrene of the right upper limb extended upto axilla. He also had electrical burn injury over the left upper thigh and penis. Disarticulation was done at the right shoulder level along with wound debridement over the left upper thigh and penis. Immediate reconstruction could not be done as the general condition of the patient was not good. Wound debridement was repeated after 2 days and reconstructed with tensor fascia lata flap. Total amputation of the penis was done as it became gangrenous due to progressive tissue necrosis.

Various studies[34,45,46,49] showed that early would debridement and flap cover produced good results. In most of the studies the average total body surface area involvement was 15.2%.

A study by Handschin AE et al showed that average of 4.8 ± 4 operations were performed per patient (range 1 – 23 operation) in a total of 61 patients. This retrospective study was conducted between 1995-2007 surgical procedures included repeated debridements (100% of all patients), early escharotomy/fasciotomy (47.5%) and amputations 18% (14 patients), 23% underwent reconstructive surgery using either local or free flaps. Mortality rate was 15%.

Our study included 52 cases. The mean total burn area was 25%. In our study, fasciotomies were done in 28% of cases (15 cases), amputations which excluded the disarticulation of fingers were 19% (10 cases). Disarticulation of the fingers were done in 8 cases including 2 thumbs pedicled distant flaps were done in 15 cases (28.8%) and split thickness skin graft was done in 19 cases (36.5%). In our study mortality rate was 3.8% (2 cases).

COMPARATIVE ANALYSIS

S. No.	Studies	Total no. of patients	Fasciotomies	Ampu-tations	Wound debridement and early flap cover	Serial excisions and delayed reconstruction	Mortality rate	Complication
1.	Liu, HY et al., 1989	121	12%	42%	35 predicted flaps		4%	Flap loss in 3 cases
2.	Wang, H et al., 2000	62	32%	45%	29 Groin flaps		7%	Partial loss of flap in 1 case
3.	Zhu, Z et al., 2000	105	27%	7%	20 flaps		8%	1 flap loss
4.	Handschin, AE et al., 2007	61	47%	18%	14 patients	4 patients	15%	Wound infection in 4 cases
5.	This study	52	15 cases 28.8%	18 cases 34.6%	12 pedicled flaps (75%)	3 pedicled flaps (25%)	3.8%	Partial loss of flap in 1 case and infection in 3 cases.

CONCLUSION

A study of management of acute electrical burns of upper limbs was done in 52 cases in our institution in a period of 2½ yrs and the following conclusions were drawn.

1. Early fasciotomy, repeated debridements, non postponement of definitive skin cover (SSG/flap) have helped in reducing the morbidity and improving the quality of life of the patient apart from reducing total hospital stay.

2. The key of success still remains in meticulous debridement of the non-viable tissues, thereby preparing the bed for early skin cover.

3. Pedicled distant flaps are reliable, durable and easy to perform for the post electrical burn defects of the upper limbs, which gives predictable results.

4. Pre-burn conditions such as anemia, greatly influence the final outcome.

5. Early wound debridement and skin cover has helped in reduction in the rate of amputations and faster wound healing. However reconstruction by flap cover has been delayed in some cases because of the poor general condition of the patients and the presence of extensive injuries over various regions of the body.

MASTER CHART

Sl. No.	Name	Age/ Sex	IP No.	Diagnosis	Procedures done after admission (PBD)						
					I	II	V	VII	1-2 wks	2-3 wks	Complications
1.	Mookan	47/M	25143	28% PEB Gangrene both UL-Distal forearm			BIL. BE Amputation				Healed/stump – prosthesis given
2.	Sukumar	20/M	26027	30% PEBRA Both UL & gangrene (R) thumb	Fasciotomy		Thumb disarticulation + Groin flap				Flap heated well
3.	Shankar	21/M	26261	30% PEBRA Both UL						WD + SSG	Wound healed well
4.	Venkatesh	20/M	26026	27% PEBRA Both UL					WD + SSG		Wound healed well
5.	Arul	28/M	26028	30% PEB Gangrene (R) UL		Fasciotomy	(R) BE Amputation				Healed stump + prosthesis given
6.	Sathish Kumar	14/M	27578	PEB Gangrene Both UL	Fasciotomy forearm & hand			BIL. BE Amputation			Healed stump + prosthesis given
7.	Subramani	40/M	26010	PEBRA (R) UL & scapula		Fasciotomy forearm	Wound debridement		SSG		Wound infection Px by antibiotics
8.	Sukumar	20/M	26027	PEBRA (R) UL wrist & thigh	Fasciotomy			WD + Bilobed groin flap	SSG		Partial loss of flap – managed by SSG
9.	Venkatesan	23/M	26026	PEBRA both UL & LL					BE (R) amputation		
10.	Asish	28/M	27514	20% EB + PEB Gangrene (R) index & thumb		Fasciotomy hand & forearm		Disarticulation of thumb + Groin flap			

Sl. No.	Name	Age/ Sex	IP No.	Diagnosis	Procedures done after admission (PBD)						
					I	II	V	VII	1-2 wks	2-3 wks	Complications
1.	Mookan	47/M	25143	28% PEB Gangrene both UL-Distal forearm			BIL. BE Amputation				Healed/stump – prosthesis given
11.	Rajendran	32/M	27540	PEBRA palm of the (R) hand	Fasciotomy of hand		WD	Superiorly based abdominal flap			
12.	Senthilvelan	27/M	27601	PEBRA both UL					WD + SSG		
13.	Rajan	30/M	27625	PEB injury (L) wrist & distal forearm					WD + Bilobed groin flap		
14.	Kuppan	32/M	27645	18% PEBRA with volar aspect forearm defect		Fasciotomy forearem		WD	Superioly based abd. flap		
15.	Sudakar	26/M	27682	25% PEB Defect I web (R) hand	Fasciotomy hand			WD	Groin flap		
16.	Anandan	35/M	28421	15% PEB Eschar				SSG			
17.	Sundar	41/M	28706	27% PEBRA				SSG			
18.	Mani	19/M	28909	30% PEB Gangrene both UL	Fasciotomy forearm & hand			BIL. BE amputation	Secondary hemorrhage		Secondary hemorrhage on 12th PEBD proximal ligation & Revision amputation was done
19.	Ravi	20/M	29413	25% PEB Defect (R) hand over thumb				WD	PIA flap		
20.	Rajesh	25/M	30701	PBE schar (R) UL				SSG			
21.	Shanmugam	44/M	31425	PEBRA (L) hand		Fasciotomy			Disarticulati		

Sl. No.	Name	Age/ Sex	IP No.	Diagnosis	Procedures done after admission (PBD)						
					I	II	V	VII	1-2 wks	2-3 wks	Complications
1.	Mookan	47/M	25143	28% PEB Gangrene both UL-Distal forearm			BIL. BE Amputation				Healed/stump – prosthesis given
						(L) hand			on of ring & little finger		
22.	Kulsan	24/M	31370	PEB Gangrene (R) forearm				BE amputation (R) UL			
23.	Eswaraiah	25/M	10574	PE Gangrene (R) hand		Fasciotomy (R) hand			Disarticu-lation of (R) index & mid finger		
24.	Chandran	43/M	11721	PEB Gangrene (R) thumb			WD		WD	Groin flap for thumb reconstruction	
25.	Manimaran	28/M	12607	PEB Gangrene (R) UL	Fasciotomy (R) forearm & hand			Disarticulation of (R) shoulder			
26.	Govindan	42/M	12702	PEBRA(L) arm			WD + collagen		WD + SSG		
27.	Jayaraj	46/M	13402	PEBRA Dorsum of the (L) hand		Fasciotomy (L) hand		WD	Inferiorly based abd. flap		Flap was healthy wound infection was treated with metronidazole & cotrimoxazole
28.	Anandan	35/M	13501	PEBRA (L) arm						SSG	
29.	Nagaraj	20/M	13692	PEBRA (R) Axilla				SSG			
30.	Stanley	27/M	14421	PEBRA (R) arm				SSG			

Sl. No.	Name	Age/ Sex	IP No.	Diagnosis	Procedures done after admission (PBD)						
					I	II	V	VII	1-2 wks	2-3 wks	Complications
1.	Mookan	47/M	25143	28% PEB Gangrene both UL-Distal forearm			BIL. BE Amputation				Healed/stump – prosthesis given
	Devakumar										
31.	Surendar	18/M	14723	PEB Gangrene (L) thumb		Fasciotomy (L) hand			Groin flap		
32.	Kumar	20/M	14951	PEB Gangrene (R) little finger	Fasciotomy (R) hand			Disarticulation (R) little finger			
33.	Nagendren	41/M	15072	PEBRA (L) arm						SSG	
34.	Anbarasu	26/M	15120	PEBRA both elbow				SSG			
35.	Sarathy	25/M	16271	30% EB with PEB Gangrene both UL & LL & trunk	Pt expired						
36.	Deva	24/M	17412	PEBRA (R) band		Fasciotomy (R) hand			WD	SSG	
37.	Raju	25/M	18562	PEBRA (L) wrist	Fasciotomy (L) hand					SSG	
38.	Prabhu	23/M	15270	PEBRA (R) wrist		Fasciotomy (R) wrist & forearm				SSG	
39.	Muthu	27/M	15310	PEB Defect I web space (R) hand				WD	Groin flap		Wound infection was controlled by antibiotic (Amikacin)
40.	Mohamed	26/M	15390	PEBRA (R)			WD		WD + SSG		

Sl. No.	Name	Age/ Sex	IP No.	Diagnosis	Procedures done after admission (PBD)						
					I	II	V	VII	1-2 wks	2-3 wks	Complications
1.	Mookan	47/M	25143	28% PEB Gangrene both UL-Distal forearm			BIL. BE Amputation				Healed/stump – prosthesis given
				forearm							
41.	Srinivasan	32/M	15408	PEBRA (L) elbow region			WD + collagen		SSG		
42.	Murali	21/M	15421	PEBRA (L) forearm & hand				Disarticulation of (R) index			
43.	Tangarasu	23/M	15671	PEBRA (R) hand Dorsm	Fasciotomy (R) hand			Disarticulation of index & mid finger			Wound infection was treated with metronidazole & cefotaxim
44.	Nanthini	6/F	16205	PEB Gangrene of (R) little finger				Disarticulation of (R) little finger			
45.	Anjaneyalu	18/M	16705	PEBRA (L) forearm & hand		Fasciotomy (L) hand		Disarticulation of thumb + Groin flap			
46.	Dayalan	14/M	16871	PEB Defect (R) hand Dorsum						Reverse radial forearm flap	
47.	Surendar	24/M	17080	30% EB + PEB Gangrene both UL, trunk & LL			Pt expired				Pt. expired due to renal failure
48.	Sampath	35/M	17545	PEB Gangrene (R) hand & Distal forearm				BE amputation (R) UL			Stump wound infection was treated wtih gentamycin & metronidazole

Sl. No.	Name	Age/ Sex	IP No.	Diagnosis	Procedures done after admission (PBD)						
					I	II	V	VII	1-2 wks	2-3 wks	Complications
1.	Mookan	47/M	25143	28% PEB Gangrene both UL-Distal forearm			BIL. BE Amputation				Healed/stump – prosthesis given
49.	Sivakumar	28/M	18476	PEBRA (L) wrist					Groin flap		
50.	Prabahar	20/M	19165	PEB (R) hand				WD + Groin flap			
51.	Kumar	35/M	20435	PEB Defect (L) forearm		Fasciotomy (L)forearm			WD + collagen	Inferiorly based abd. flap	
52.	Boopalan	41/M	20708	PEBRA (R) UL				WD + SSG			

BIBLIOGRAPHY

1. Richard F Edlich et al. Article on Electrical burns, University of Virginia-e.medicine.

2. Luce E.A. Electrical injuries. In: J.G. McCarthy (Ed.), 'Plastic Surgery', 814-830, WB Saunders Company.

3. Brain E Benson, MD et al. Article on burns, Electrical injury, e-medicine.

4. James M. Shaw and Martin et al. Electrical injuries total burn care, 401-406.

5. Lee RC. Injury by electrical forces: pathophysiology, and manifestation. Curr. Probl. Surg. Sep.1997; 34(9): 677-764.

6. Leibovici D. Shemer J. Shapira SC, Electrical injuries: Current concepts. Nov.1995; 26(9): 623-7.

7. Skoog T. Electrical injuries. J. Trauma 1970; 10: 816-30.

8. Robson Mc Morphy RC. Heggers JP. A new explanation for the progressive tissue loss in electrical injury. PRS 1984; 73 : 431-7.

9. Hunt JL, Mason AD Jr, Masterson TS et al. The pathophysiology of electrical injuries. J. Trauma May 1976;l 16(5): 335-340.

10. Baxter CR. Present concepts in management of major electrical injuries. Surg. Clin. North Am. 1970; 50 : 1401-18.

11. Luce EA et al. True high tension electric injuries. Ann. Plast. Surg. 12 : 321-326, 1984.

12. Wilkinson C. Wood M. High voltage electric injury. Am. J. Surg., 136: 693-696, 1984.

13. Ponten B. Erikson U et al. New observation on tissue changes along the pathway of the current in an electrical injury. Scant J. Plast. Recon. Sng. 1970; 4 : 75-80.

14. Edwards FW. Compartment syndrome. J. Bone Joint Surg. 1968; 518 : 123-125.

15. Levine NS, Atkins A. et al, Spinal cord injuries following electrical accidents; case reports. J. Trauma, May 1975; 15(5): 659-63.

16. Hunt JL et al. Vascular lesions in acute electrical injuries. J. Trauma 1974; 14 : 461-73.

17. Baxter CR. Present concepts in management of major electrical injuries. Surg. Clin. North Am. 1970; 1401-18.

18. Robson MC et al. Immediate and delayed cellular damage following soft tissue. Essays in surgery, New York, Churchill Livingstone. 1989; 153-8.

19. Robson MC et al. Role of arachidonic acid metabolism in electrical injury. Electrical trauma: Cambridge University Press, 1992; 179-88.

20. Naidu SI et al. Compartment syndrome, a surgical emergency. Am. Surt. May 1977; 43(5) : 303-9.

21. Brain E. Benson et al. Article on electrical burns, e-medicine.

22. Colic M, Ristic L et al. Emergency treatment and early fluid resuscitation following electrical injuries. Acta Chir. Plast. 1996; 38(4): 137-41.

23. Koffler A et al. Acute renal failure one to non traumatic rhabdomyolysis. Ann. Intern. Med. Jul. 1976; 85(1) : 23-8.

24. Chris Oliver et al. Compartment syndromes of the forearm and hand, University of Edinburg.

25. Sheridar GW, Matsen FA 3d, Fasciotomy in the management of acute compartment syndrome. J. Bone Joint Surg. Jan. 1976; 58(1): 112-5.

26. Edlich RF et al. Technical considerations for fasciotomies in high voltage electrical injuries. J. Born Care Rehabil., 1980; 1 : 22-25.

27. Ponten B et al. New observation on tissue changes along the pathway of the current in electrical injury. Scand J. Plast Recon. Surg., 1970; 4 : 75-80.

28. Hunt J et al. Acute electric burns. Current diagnostic and therapeutic approaches to management. Arch. Surg. Apr. 1980; 115(4) : 434-8.

29. Clayton JM et al. Xenon-133 determination of muscle blood flow in electrical injury. J. Trauma 1977; 17 : 293-8.

30. Zelt RC et al. Electrical injury in: Marsh J ed, Current therapy in plastic and recon. Surg. 1989; 29-33.

31. Penneys NS. Prostaglandins and the skin. Current concepts. The Upjohn Co. 1980; 34-9.

32. Li YY, Wang JL et al. Resurfacing upper extremity wounds with predicted groin flaps. Burns, 2000 May, 26(3): 283-8.

33. Baumeister S. et al. Principles of microvascular reconstruction in electrical burn wounds. Burns 2005; 31 : 92-8.

34. Liu HY et al. Experiences in the treatment of electrical born covering deep wounds with flaps.

35. Wilkinson C et al. High voltage electric injury. Am. J. Surg., 136 : 693-696, 1978.

36. Arturson G et al. Fluid resuscitation. Scand. J. Plast. Recon. Surg, 18 : 111-118, 1984.

37. Rouse RG et al. A review of pathophysiology and comparison of patient management protocols. J. Trauma, 18 : 43-47, 1978.

38. Yowler CJ et al. Factors contributing extremity amputation in burn patients. J. Trauma., Sep.1998; 45(3): 522-6.

39. Holliman CJ et al. Early surgical decompression in the management of electrical injuries. Am. J. Surg., 144 : 733-739, 1982.

40. Parshley PF et al. Aggressive approach to the extremity damaged by electric current. Am. J. Surg. 150 : 78-82, 1985.

41. Hunt JL et al. Pathophysiology of acute electrical injuries. J. Trauma., 1976; 16 : 335-40.

42. Loce EA et al. High-tension electrical injury of the upper extremity. Surg. 147: 38-42, 1978.

43. Govila A. Early excision and primary resurfacing of wounds following high voltage electrical burns. Eur. J. Plast. Surg., 12 : 147-154, 1989.

44. Sturim HS. The treatment of electrical injuries. J. Trauma, 1971; 11 : 959-65.

45. Wang XW et al. Early skin grafting for electrical injuries. J. Trauma, 26 : 128-134, 1986.

46. Zhuz et al. Analysis of urgent reconstruction of electrical injuries. Chin J. Traumatol., 2000, Nov.15; 3(4) : 214-218.

47. Silverberg B et al. Microsurgical reconstruction for the electrical injury. Proc. Am. Burn. Assoc., 1985; 17 : 129.

48. Zelt RG et al. Experimental high voltage electrical burns : role of progressive necrosis. Surg. Forum 1986; 37 : 624-6.

49. Handschin AE et al. Surgical treatment of high voltage electrical injuries. Mand Chir Mikrochir Plast Chir, 2007 Oct., 39(5): 345-9.